Amendments to the Specification

Please amend the Specification as follows:

At page 1, first sentence:

This application is a continuation-in-part of ~~USSN 09/153,198 filed September 15, 1998~~USPN 6,420,176 issued Jul. 16, 2002, which is a continuation-in-part of 60/058,933, filed September 15, 1997, both which are incorporated herein as if set forth in full.

At page 7, lines 2-17

Fig. 7a-7d. FACS analysis of cells emigrating from the skin. Plasmid DNA encoding the Green Fluorescent Protein (pGFP) was used as a reporter gene: 7a) PEIm/DNA complex applied on the surface of the skin; 7b) Control skin; 7c) PEIm/DNA complex injected subcutaneously; 7d) FITC-dextran injected subcutaneously.

Fig. 8a-8c. DNA-modified cells in the lymph node of mice after transcutaneous DNA immunization: 8a) Transduced cells expressing plasmid DNA-derived gene entering into the lymph node detected by in situ hybridization (white silver grains over the cells) labeled by the antisense Neo probe; 8b) Enlargement of Fig. 8a; 8c) Immunohistochemical staining of a lymph node to detect protein expressing cells.

Fig. 9a-9b. Dendritic cells expressed the plasmid DNA in a macaque's lymph node following transcutaneous DNA immunization: 9a) In situ hybridization dark-field microscopic image of cells showing (white) silver grains over positive mononuclear cells at the periphery of a lymph node; 9b) A single DNA expressing cell stained with p55 (brown) that is a marker for lymph node DC. The black dots are silver grains (in situ hybridization) demonstrating the expression of the foreign gene.

At page 7, lines 26-30

Fig. 11a-11b. Median viral load and CD4 counts during HAART (11a) and STI-HAART (11b) treatment of SIV251-infected rhesus macaques with AIDS. Monkeys were treated with d(R)-9-(2-Phosphonylmethoxypropyl) adenine, didanosine and hydroxyurea

("HAART") during the indicated time, as described previously 1. Symbols: triangles, CD4 counts; squares, viral load.

At page 8, lines 1-6

Fig. 12a-12b. DermaVirSHIV vaccination in combination with STI-HAART for the treatment of SIV251-infected macaques with AIDS. Treatment schedule and median viral load of the cohort before (12a) and after (12b) the initiation of therapeutic vaccination.

Fig. 13a-13b. Virological and immunological characterization of monkey #51 (13a), #56(13b), #60 (13c) treated with STI-HAART and DermaVirSHIV. Treatment: dotted line (details see Fig. 12b). Symbols: squares, viral load; triangles, CD4 counts.

At page 21, lines 1-16

Drug combinations that are effective to at least temporarily inhibit HIV replication are known. The inventors have shown that drug combinations including hydroxyurea, one or more reverse transcriptase inhibitors and, optionally, one or more protease inhibitors are particularly effective, and, for some patients, allow the possibility of stopping drug treatment for extended periods of time. See ~~USSN 09/056,691, filed Apr. 8, 1998~~ USPNs 5,977,086 and 6,114,312 issued Sept 5, 2000 "Method of Inhibiting ~~HIV~~ Human Immunodeficiency Virus by Combined Use of Hydroxyurea, a Nucleoside Analog, and a Protease Inhibitor", ~~USSN 09/048,753 filed Mar. 26, 1998~~USPN 6,251,874 issued Jun 26, 2001, "Method of Inhibiting ~~HIV~~ Human Immunodeficiency Virus using Hydroxyurea and Reverse Transcriptase Inhibitor in vi~~tro~~" and USSN ~~09/048,756~~09/048,576, filed March 26, 1998, "Method of Rendering a HIV Population Replication Incompetent in vivo", abandoned, all of which are incorporated herein by reference as if set forth in full. The present invention includes the treatment of a patient with active HIV infection with an appropriate drug combination until the viral load in the blood has been effectively suppressed, that is, has reached a suitably low level, less than about 50,000 copies per ml, preferably less than 10,000 copies per ml, more preferably

less than 200-500 copies per ml. The patient is then vaccinated using the present invention while the drug combination suppresses replication of the wild-type virus.

At page 22-23, the bridging paragraph

Reverse transcriptase inhibitors figure prominently in current HIV treatments. Examples include nucleoside analogs, such as the 2',3'-dideoxyinosine (ddI)(available as Videx® from Bristol Myers-Squibb). Nucleoside analogs are a class of compoounds known to inhibit HIV, and ddI is one of a handful of agents that have received formal approval in the United States for clinical use in the treatment of AIDS. Like zidovudine (3'-azido-2',3' -dideoxythymidine or azidothymidine [AZT] available from Glaxo Wellcome), zalcitabine (2',3' - dideoxycytidine [ddC] available as Hivid® from Hoffman-La Roche), lamivudine 2'-deoxy-3'-thiacytidine [3TC](Epivir® available from Glaxo Wellcome), Iodenosine (F-ddA available from US Biosciences and stavudine (2',3' -didehydro-2',3'-dideoxythimidine [D4T] available as Zerit® from Bristol Myers-Squibb), ddI belongs to the class of compounds known as 2',3' - dideoxynucleoside analogs, which, with some exceptions such as 2',3'-dideoxyuridine [DDU], are known to inhibit HIV replication, but have not been reported to clear any individual of the virus. Other nucleoside reverse transcriptase inhibitors include adefovir dipivoxil [PMEA], or Preveon® an adenine nucleotide analog from Gilead Sciences), abacavir (1592U89 available as Ziagen® from ~~Glaxo Wellcome~~GlaxoSmithKline), ~~lubocavir (a guanosine analog available from Bristol Meyers Squibb),~~ and tenofovir DF, [PMPA], available as Viread® from Gilead ~~Pharmaceuticals~~ Sciences Inc. New nucleosides include emtricitabine, [FTC] ~~(Emtricitabine)~~, available as Emtriva® from Gilead Sciences, amdoxovir, [DAPD, also known as DXG] available from Gilead Sciences, F-ddA (Lodenosine, a fluorinated purine nucleoside RTI, and dOTC (BCH-10562). Non-nucleoside reverse transcription inhibitors include nevirapine (Viramune™ available from Boehringer Ingelheim Pharmaceuticals, Inc.), delaviridine (Rescriptor® available from Pharmacia & Upjohn) and efavirenz (available as Sustiva®, from ~~DuPont Merck~~Bristol-Myers Squibb)

At page 23-24, the bridging paragraph

Of the potential protease inhibitors for use against HIV, compounds such as hydroxyethylamine derivatives, hydroxyethylene derivatives, (hydroxyethyl)urea derivatives, norstantine derivatives, symmetric dihydroxyethylene derivatives, and other dihydroxyethylene derivatives have been suggested, along with protease inhibitors containing the dihydroxyethylene transition state isostere and its derivatives having various novel and high-affinity ligands at the P2 position, including 3-tetrahydrofuran and pyran urethanes, cyclic sulfolanes and tetrahydrofuranylglucines, as well as the P3 position, including pyrazine amides. In addition, constrained "reduced amide"-type inhibitors have been constructed in which three amino acid residues of the polypeptide chain were locked into a g-turn conformation and designated g-turn mimetics. Other alternatives include penicillin-derived compounds and non-peptide cyclic ureas. Sßuitable protease inhibitors include Indinavir sulfate, (available as CrixivanTM capsules from Merck & Co., Inc, West Point, PA.), saquinavir (Invirase® and Fortovase® available from Hoffman-LaRoche), ritonavir (Norvir® available from Abott Laboratories) ABT-378 (available from Abott Laboratories), Nelfinavir (Viracept® from Agouron Pharmaceuticals Inc), ~~and GW141 (available from Glaxo Wellcome/Vertex)~~ Tipranavir available from Boehringer Ingelheim GmbH~~Pharmacia & Upjohn~~, PD 178390 available from Parke-Davis, atazanavir [BMS-23632] available as Reyataz® ~~available~~ from Bristol-Myers Squibb), DMP-450 available from Triangle, and JE 2147 available from Agouron. New protease inhibitors include ABT-378 (Abbott laboratories), L-756423, DMP-450 and AG1776.